See Article page 385.

Commentary: A dimensional deep dive

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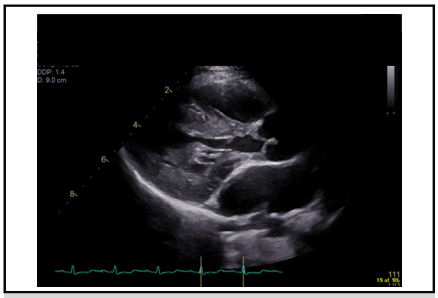
One of the anatomic hallmarks of complete atrioventricular septal defect (AVSD) is deficiency of the inlet septum with accompanying elongation of the left ventricular outflow tract (LVOT), the "goose-neck deformity." This anatomic characteristic presumably contributes to late reoperation for LVOT obstruction, which occurs in roughly 0.5% to 5% of patients.[1] The current study by Chandiramani and colleagues[2] from the Royal Hospital for Children Glasgow takes an echocardiographic deep dive into the dimensions of the LVOT in patients undergoing repair of complete AVSD. It was presented at the 2022 Annual Meeting of the American Association for Thoracic Surgery and builds on the authors' previous work, presented at the 2020 Annual Meeting of the European Association for Cardiothoracic Surgery.[3]

The current study includes a cohort of 108 patients undergoing repair of complete AVSD from 2008 to 2021. Patient characteristics are in line with current practice, with median age = 4 months, median weight = 4.8 kg, and trisomy 21 in 79%. The repair was the 2-patch technique in 67 patients (62%) and modified single-patch technique (Australian, or Nunn) in 41 patients (38%). The diameter of the LVOT was measured by echocardiography (long-axis parasternal view) prerepair and postrepair. Patients undergoing repair of isolated ventricular septal defect (VSD) served as controls. Dimensions of the aortic annulus and subaortic area were expressed as z scores and subaortic/aortic annulus ratio (with ratio disproportion defined as a ratio <0.9). The paper contains a plethora of data and comparisons. There are 3 primary findings:



LVOT obstruction postrepair of AVSD.

CENTRAL MESSAGE

Surgical repair of complete AVSD results in a disproportionate decrease in the dimension of the subaortic area, particularly with use of the modified single patch technique.

1. Prerepair: AVSD aortic annulus and subaortic areas were similar in size (ratio = 1.0), but both were smaller than in patients with VSD.
2. Postrepair: AVSD showed a disproportionate decrease in the subaortic area, with a decrease of both subaortic z score (from −0.5 to −0.9) and subaortic/aortic annulus ratio (from 1.0 to 0.95). In contrast, the dimensions in patients with VSD did not change from prerepair.
3. Postrepair: the disproportionate decrease in AVSD subaortic area was more pronounced in patients who received a modified single-patch operation than in patients who received a 2-patch operation; postrepair, 41% of modified patients who received a modified single-patch operation had a subaortic z score <−2.0 (vs 12% of patients who received 2-patch), whereas 51% had disproportion of the subaortic/aortic annulus ratio (vs 37% of patients who received 2-patch).

In summary, LVOT dimensions in a complete AVSD start out smaller than those in a VSD, and AVSD repair disproportionally decreases the dimension of the subaortic area, particularly when a modified single-patch repair is used. These findings beg 2 questions.

First, is the more-pronounced effect of the modified single-patch repair on the subaortic area a result of the technique itself, which pulls the atrioventricular valve onto the crest of the septum, and potentially into the LVOT? Or, is this finding attributable to the selection of patients with a shallower VSD for the modified single-patch repair?

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Prerepair, the modified single-patch group had a median subaortic z score = −0.7, with 10% being <−2.0, compared with the 2-patch group, with a median subaortic z score = −0.4% and 4% being <−2.0. Prerepair subaortic/aortic annulus ratio disproportion (<0.9) was seen in 12% of patients in both groups. These similarities prerepair indicate that the repair technique may play the larger role. A comparison of the 2 techniques with patients matched for prerepair depth of VSD would be needed to give a more definitive answer.

Second, do these findings imply that the modified single-patch technique will have a greater incidence of late LVOT obstruction requiring reoperation? In the current study, the answer was no, but only 4 patients required LVOT reoperation at a mean follow-up of 6.8 years. Several other series have documented a low rate of LVOT reoperation after modified single-patch repair.[4,5] A 4-center Australian series of 819 patients found an LVOT reoperation rate = 3.8%, with no difference between the modified single-patch and 2-patch technique, even when the patients were propensity matched (although they were not matched for depth of VSD).[6] The LVOT after repair of AVSD is complex, and LVOT dimension is likely only one factor determining late obstruction (Figure 1). Other factors may include the anterolateral muscle bundle of the left ventricle, abnormal attachment of the superior bridging leaflet in the LVOT, abnormal positioning of the atrioventricular subvalvular apparatus, accessory atrioventricular valve tissue, fibromuscular obstruction, and septal hypertrophy.[1] As Dr Van Arsdell[7] pointed out more than 25 years ago, the consequence is that when LVOT obstruction occurs, "solutions may be complex and palliative."

A couple of limitations to this study should be noted. The LVOT was measured in one dimension only; imaging of cross-sectional area may paint a different picture. Also, the study examines the structure, but not the function, of the LVOT. It would be of interest to look at whether any of these patients developed postrepair LVOT gradients and how these correlated with LVOT dimensions. Nonetheless, I commend the authors for their echocardiographic expertise and the clarity of their analysis and presentation.

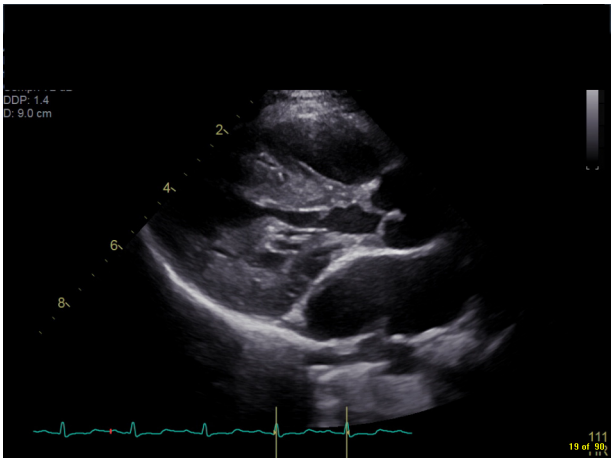

FIGURE 1. Left ventricular outflow tract obstruction 3 years following repair of AVSD. The obstruction was caused by a combination of fibromuscular hypertrophy and anomalous left atrioventricular valve chordal support.

This dimensional deep dive may not change our surgical approach, but it has given us accurate and interesting new information on the LVOT in patients with complete AVSD.

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